

of this notice is to allow an additional 30 days for public comment. The National Institutes of Health may not conduct or sponsor, and the respondent is not required to, an information collection that has been extended, revised, or implemented on or after October 1, 1995, unless it displays a currently valid OMB control number.

*Proposed Collection:* Title: HIV Vaccine Awareness Study-Americans' Attitudes. *Type of Information Collection Request:* New. *Need and Use*

*of Information Collection:* NIH/NIAID/DAIDS is in the process of planning a campaign to inform Americans about HIV preventive vaccine research. As part of planning, it is necessary to establish a baseline of Americans' levels of knowledge and attitudes with respect to HIV preventive vaccine research; to determine what information is required by communities to address the mistrust, myths, and misinformation about HIV vaccine research; and to identify how and what information should be

provided to communities to promote more positive attitudes toward HIV vaccine research. Findings will help inform initial campaign decisions and serve to evaluate the effectiveness of the campaign's efforts. *Frequency of Response:* Two times. *Affected Public:* Individuals or households. *Type of Respondents:* Random samples of adults, including those considered at-risk for HIV and members of their social networks. The annual reporting burden is as follows:

Type of respondents	Estimated number of respondents	Estimated number of responses per respondent	Average burden hours per response	Estimated total annual burden hours requested
General Population Adults .....	4,000	1	.25	1,000
HIV-Affected Adults .....	3,000	1	.25	750
Total .....	7,000	.....	.25	1,750

The annualized cost to respondents is estimated at \$17,500. There are no Capital Costs to report. There are no Operating or Maintenance Costs to report.

*Request for Comments:* Written comments and/or suggestions from the public and affected agencies are invited on one or more of the following points: (1) Whether the proposed collection of information is necessary for the proper performance of the function of the agency, including whether the information will have practical utility; (2) The accuracy of the agency's estimate of the burden of the proposed collection of information, including the validity of the methodology and assumptions used; (3) Ways to enhance the quality, utility, and clarity of the information to be collected; and (4) Ways to minimize the burden of the collection of information on those who are to respond, including the use of appropriate automated, electronic, mechanical, or other technological collection techniques or other forms of information technology.

*Direct Comments to OMB:* Written comments and/or suggestions regarding the item(s) contained in this notice, especially regarding the estimated public burden and associated response time, should be directed to the: Office of Management and Budget, Office of Regulatory Affairs, New Executive Office Building, Room 10235, Washington, DC 20503, Attention: Desk Officer for NIH. To request more information on the proposed project or to obtain a copy of the data collection plans and instruments, contact Robert J. Gulakowski, Health Sciences Communications Specialist, DAIDS,

NIAID, NIH, 6700-B Rockledge Drive, MSC 7620, Room 4144, Bethesda, MD 20892-7620, or call non-toll free (301) 496-0545, or E-mail your request, including your address to [rg106x@nih.gov](mailto:rg106x@nih.gov).

*Comments Due Date:* Comments regarding this information collection are best assured of having their full effect if received within 30-days of the date of this publication.

Dated: July 31, 2002.

**Brenda J. Velez,**

*Chief, CMB, NIAID and NIAID Project Clearance Liaison.*

[FR Doc. 02-19868 Filed 8-6-02; 8:45 am]

**BILLING CODE 4140-01-M**

**DEPARTMENT OF HEALTH AND HUMAN SERVICES**

**National Institutes of Health**

**Government-Owned Inventions; Availability for Licensing**

**AGENCY:** National Institutes of Health, Public Health Service, DHHS.

**ACTION:** Notice.

**SUMMARY:** The inventions listed below are owned by agencies of the U.S. Government and are available for licensing in the U.S. in accordance with 35 U.S.C. 207 to achieve expeditious commercialization of results of federally-funded research and development. Foreign patent applications are filed on selected inventions to extend market coverage for companies and may also be available for licensing.

**ADDRESSES:** Licensing information and copies of the U.S. patent applications

listed below may be obtained by writing to the indicated licensing contact at the Office of Technology Transfer, National Institutes of Health, 6011 Executive Boulevard, Suite 325, Rockville, Maryland 20852-3804; telephone: 301/496-7057; fax: 301/402-0220. A signed Confidential Disclosure Agreement will be required to receive copies of the patent applications.

**An Obligate Domain-Swapped Dimer of Cyanovirin with Enhanced Anti-Viral Activity**

Carole A. Bewley and Brendans Kelly (NIDDK).

DHHS Reference No. E-096-02/0 filed 25 Feb 2002.

*Licensing Contact:* Sally Hu; 301/496-7056 ext. 265; e-mail: [hush@od.nih.gov](mailto:hush@od.nih.gov).

The present invention provides a purified or isolated obligate domain-swapped dimer of Cyanovirin-N (CVN hereafter), a method of making an obligate domain-swapped dimer of CVN and a method of inhibiting a viral infection of a mammal by administering domain-swapped dimer of CVN. CVN is outstanding in that it potently blocks viral entry in all human and simian isolates by binding to HIV through highly avid and very specific carbohydrate-mediated interactions with the surface envelope glycoprotein gp120. CVN has also been shown to form a domain-swapped dimer under non-physiological conditions such as mM concentration and low pH. This invention provides an obligate domain-swapped dimeric mutant of CVN, called ΔQ50-CVN, which has several significant advantages over the wild-type CVN: First, ΔQ50-CVN can be purified from a crude bacterial cell

lysate in a single chromatographic step because it forms only one homogeneous species. Second, because this obligate dimer has four carbohydrate binding sites, it binds gp120 and other glycoproteins with greater affinity than wild-type CVN. Third, ΔQ50–CVN shows an enhancing increase in efficacy in blocking viral entry in a quantitative HIV–1 envelope-mediated cell fusion assay. Thus, ΔQ50–CVN displays enhanced anti-HIV activity relative to the wild-type CVN monomer and offers a great advantage over wild-type CVN because it is extremely easy to purify large quantities to greater than 95% homogeneity. So, it may open the possibility that an effective drug treatment for HIV could reach underdeveloped countries.

Finally, the background of this invention is further described in J. Am. Chem. Soc. (2002) 124:3210–3211, J. Magn. Reson. (2002) 154:329–335, Structure (2001) 10:931–940, J. Am. Chem. Soc. (2001) 123:3892–3902, J. Am. Chem. Soc. 122: 6009–6016, and J. Mol. Biol. (1999) 288:403–412.

#### Methods and Compositions for Antagonizing Septic Shock

George Kunos (NIAAA).  
DHHS Reference No. E–321–01/0 filed  
15 Aug 2001.

*Licensing Contact:* Norbert Pontzer; 301/496–7736, ext. 284; e-mail: np59n@nih.gov.

Septic shock is an often fatal type of vasodilatory shock that may accompany microbial infections. Septic shock has therefore been an increasing problem in recent years because of the increasing number of individuals who are immunocompromised. Recent studies have indicated that the hypotension associated with hemorrhagic shock (Wagner *et al.*, Nature 1997; 390:518–521) or septic shock (Varga *et al.*, FASEB J. 1998; 12:1035–1044) may be mediated by macrophage-derived endogenous cannabinoids such as anandamide, acting at vascular cannabinoid receptors. In an earlier study (PNAS, 1999; 96:14136) the NIH inventor(s) presented several lines of evidence indicating the vasodilator effect of anandamide is mediated by a receptor distinct from the two known cannabinoid receptors, CB1 and CB2. In particular, anandamide-induced vasodilation persists in mice deficient in both CB1 and CB2 receptors. They postulated that a yet unidentified cannabinoid receptor was responsible for the observed effect. The invention described and claimed in the pending patent application provides compounds acting as agonists and antagonists at the newly described cannabinoid receptor

and methods for reversing pathological vasodilation of blood vessels observed during conditions such as septic shock.

#### Methods of Diagnosing and Treating Schizophrenia

Daniel R. Weinberger *et al.* (NIMH).  
DHHS Reference No. E–247–01/0 filed  
31 Aug 2001.

*Licensing Contact:* Norbert Pontzer; 301/496–7736, ext. 284; e-mail: np59n@nih.gov.

Neurotrophins promote survival of neurons from both the central nervous system and peripheral nervous system in cell culture. More recently it has been shown that neurotrophins may serve as a new class of neuromodulators that mediate activity-dependent modifications of neuronal connectivity and synaptic efficacy. Brain derived neurotrophic factor (BDNF) is a neurotrophin that mediates LTP and hippocampus-related spatial memory. Schizophrenia and other mental disorders appear to involve deficits in verbal memory and reduced hippocampal—acetyl aspartate (NAA), a measure of hippocampal neuronal integrity. BDNF may thus play a role in memory function and human diseases of the hippocampus such as schizophrenia.

The human BDNF gene contains one known non-conservative SNP, producing a met66val substitution. The invention is related to the discovery that a met66val polymorphism in the gene for BDNF is correlated with verbal memory and risk for schizophrenia. The invention provides methods and kits for diagnosing and modulating verbal memory and risk for schizophrenia in an individual by determining the individual's BDNF genotype, and associating a met allele with impaired verbal memory and risk for schizophrenia and a val allele with enhanced verbal memory and protection from schizophrenia. The invention also provides methods of finding and using compounds which modulate BDNF function in order to treat human diseases of the hippocampus such as memory disorders and schizophrenia.

#### Retinoids Can Increase the Potency of Anti-Cancer Immunotoxins

You N. Wu and Richard J. Youle  
(NINDS).

U.S. Patent 5,942,230 issued August 24, 1999 and U.S. Patent 6,197,528 issued March 6, 2001.

*Licensing Contact:* Richard Rodriguez; (301) 496–7056 ext 287; e-mail: rodrigur@od.nih.gov.

A unique method of potentiating the effect of anti-cancer immunotoxins has

been developed, thus offering to significantly improve the treatment of a number of cancers as well as autoimmune diseases. Prolonged treatment of human cancers with classical methods such as radiation and chemotherapy, or a combination of both, may cause greater damage than the underlying disease because healthy tissue is often damaged along with diseased tissue. More recently, immunotherapy has emerged as a new and promising therapy for treating cancer because it employs monoclonal antibodies specific for tumor cells coupled to protein toxins. Thus, cancer cells are selectively targeted for destruction. These immunotoxins are being examined in numerous clinical trials for the treatment of cancer and autoimmune diseases. However, often the protein toxin coupled to the monoclonal antibody does not pass as readily into the cytosol of the target cell as does the native protein toxin. This invention improves the effectiveness of such immunotoxins by employing retinoic acid, which disrupts the Golgi apparatus of the target cell and increases the cytosolic routing of specific protein toxins. Also included in this invention is an in vitro method for assessing the ability of a retinoid to potentiate the activity of immunotoxins.

Dated: July 29, 2002.

#### Jack Spiegel,

Director, Division of Technology Development and Transfer, Office of Technology Transfer, National Institutes of Health.

[FR Doc. 02–19866 Filed 8–6–02; 8:45 am]

BILLING CODE 4140–01–P

## DEPARTMENT OF HEALTH AND HUMAN SERVICES

### National Institutes of Health

#### Laboratory Animal Welfare: Change in PHS Policy on Humane Care and Use of Laboratory Animals

**AGENCY:** National Institutes of Health, DHHS.

**ACTION:** Amended Policy Statement.

**SUMMARY:** The NIH is changing the PHS Policy on Humane Care and Use of Laboratory Animals (PHS Policy) to permit institutions with PHS Animal Welfare Assurances to submit verification of Institutional Animal Care and Use Committee (IACUC) approval for competing applications subsequent to peer review but prior to award.

**DATES:** This change in PHS Policy is effective as of September 1, 2002 (*i.e.*, for all applications submitted for the May-June 2003 Advisory Council dates).